JOURNAL
OF THE
ARNOLD ARBORETUM

VOLUME XVII. JANUARY, 1936 NUMBER 1

STUDIES ON THE BIOLOGY OF GYMNOSPORANGIUM GLOBOSUM FARL.

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With plates 166–175 and two text figures

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I. INTRODUCTION

SINCE the pioneer work of Farlow (1880), who ascertained the alternate hosts of *Gymnosporangium globosum* Farl., many observations on various phases of the biology of this rust have been recorded; these observations, however, do not afford a complete survey of the biology of *G. globosum*. More than one hundred suscepts have been added, since 1880, to the host list, yet observations of the writer warrant the conclusion that the number of suscepts, conservatively estimated, is more than six hundred; moreover, little information was previously available

regarding their relative susceptibility. Many aspects of the progressive development of the symptoms and signs of the diseases caused by this rust have not been described in full; the incompleteness of this knowledge becomes more evident with the increase in the number of known hosts. No definite information has been available concerning the time when the aeciospores of *G. globosum* germinate or the method by which they produce infection on the Juniperus hosts; knowledge with respect to this question is a prerequisite to any attempt to control the rust on the red cedar. Aside from an academic consideration of the biology of *G. globosum,* this rust is of increasing economic importance and is causing great damage to both the ornamental and the orchard hosts in localized areas throughout the eastern part of the United States.

These desiderata have led the writer to investigate more fully the biology of *G. globosum.* A determination of the hosts and their relative susceptibility has already been published (MacLachlan, 1935). The numerous inoculations and field observations that were conducted at that time afforded an opportunity to carry on extensive studies on other aspects of the biology of *G. globosum;* the results of these investigations have been incorporated with those of other writers and are now presented.

II. NOMENCLATURE

The following names, chronologically arranged, have been given to the rust now known as *Gymnosporangium globosum* Farl. The stage in the life cycle of the rust to which the name refers precedes the name.

III **Gymnosporangium globosum** Farl. in Bot. Gaz. **11:** 236, 239 (Sept. 1886).

I *Aecidium Crataegi* var. *Oxyacanthae* Schw. Syn. Fung. Carol. 66 (40), no. 432 (1822).

I *Caeoma Cylindrites* Lk. var. *Crataegi-punctatae* Schw. Syn. Fung. Am. Bor. (Trans. Amer. Phil. Soc. II. **4:** 294, no. 2899a. 1832).

III *Gymnosporangium fuscum* DC. var. *globosum* Farl. Gymnosporangia U. S. 18, pl. 1, f. 7–11 (1880).

III *Gymnosporangium Sabinae* (Dicks.) Wint. var. *globosum* Trel. in Trans. Wisc. Acad. **6:** 133 (29) (July 1866).

III *Gymnosporangium globosum* Farl. in Bot. Gaz. **11:** 236, 239 (Sept. 1886).

I *Roestelia lacerata y* & *z* Thaxt. ex Farl. in Bot. Gaz. **11:** 240 (Sept. 1886).

I *Gymnosporangium globosum* I Thaxt. in Rept. Conn. Exper. Sta. **14:** 98 (20) (1891).

I *Roestelia globosa* Shear, N. Y. Fungi Exsicc. no. 79 (1893). Corrected label.

III *Puccinia globosa* Kuntze, Rev. Gen. Pl. 3²: 507 (1898).
III *Roestelia globosa* III Kuntze in Bot. Centralbl. 77: 300 (15 Feb. 1899).
III *Tremella globosa* Arth. in Proc. Ind. Acad. 1900: 136 (June 1901).
I *Aecidium globosum* Farl. Bibl. Index N. Amer. Fungi, 49 (1905).
III *Aecidium globosum* Arth. in Result. Sci. Congr. Bot. Wien, 1905: 343 (1906).

The names *Gymnosporangium globosum* Farl. and *Roestelia globosa* Shear stand as the authentic names for the III and I stages, respectively. The name *Gymnosporangium globosum* Farl. is now accepted as referring to either stage of the rust and is so used throughout this presentation.

III. RANGE AND ECONOMIC IMPORTANCE OF G. GLOBOSUM

A difficulty in determining the exact range and economic importance of *G. globosum* arises due to the many reports in the literature which refer collectively to the three rusts, *G. globosum*, *G. Juniperi-virginianae*, and *G. clavipes*. Nevertheless, sufficient evidence is extant to make possible accurate determinations of both range and economic importance of *G. globosum*.

Gymnosporangium globosum is confined in its range to the eastern and central parts of United States and to the southern parts of Ontario and Quebec. In Fig. 1 the distribution by states has been plotted; the circles and dots indicate the states in which the diseases caused by this rust have been reported on the Juniperus and pomaceous hosts, respectively.

A review of the literature indicates that the prevalence of this rust is steadily increasing; several factors may be involved in this phenomenon. Jones and Bartholomew (1915) offer the suggestion that the alternate hosts, the red cedar and the wild pomaceous hosts such as crab-apples and hawthorns, multiply more rapidly in open pastures and waste cut-over lands than they did in the original forests. The orchardist and ornamentalist, as well as bringing the alternate hosts closer together, have introduced on their estates many susceptible pomaceous trees that may serve as hosts. Whether or not the rust is increasing in virulence is open to question.. The rapid increase in scientific investigation during recent years, which brings to light new host species and through the discovery of these has extended the known range of the diseases caused by this rust, may give an appearance of increased prevalence and virulence.

Gymnosporangium globosum is causing more damage in some states

than in others; this, as well, may be due to the influence of man, who is constantly bringing into close range of each other, susceptible alternate hosts of the rust. In the eastern part of New York State, for example, this rust is almost on a par with *G. Juniperi-virginianae* with respect to the damage which it is causing. Stewart (1910) reports severe infection on Kieffer pears in an orchard at Long Island. According to W. D. Mills (Haskell, 1929) *G. globosum* was unusually prevalent in Duchess and Greene counties on the foliage of pears in 1928. Thomas and Mills (1929a) consider *G. globosum* as one of the three rusts (*G.*

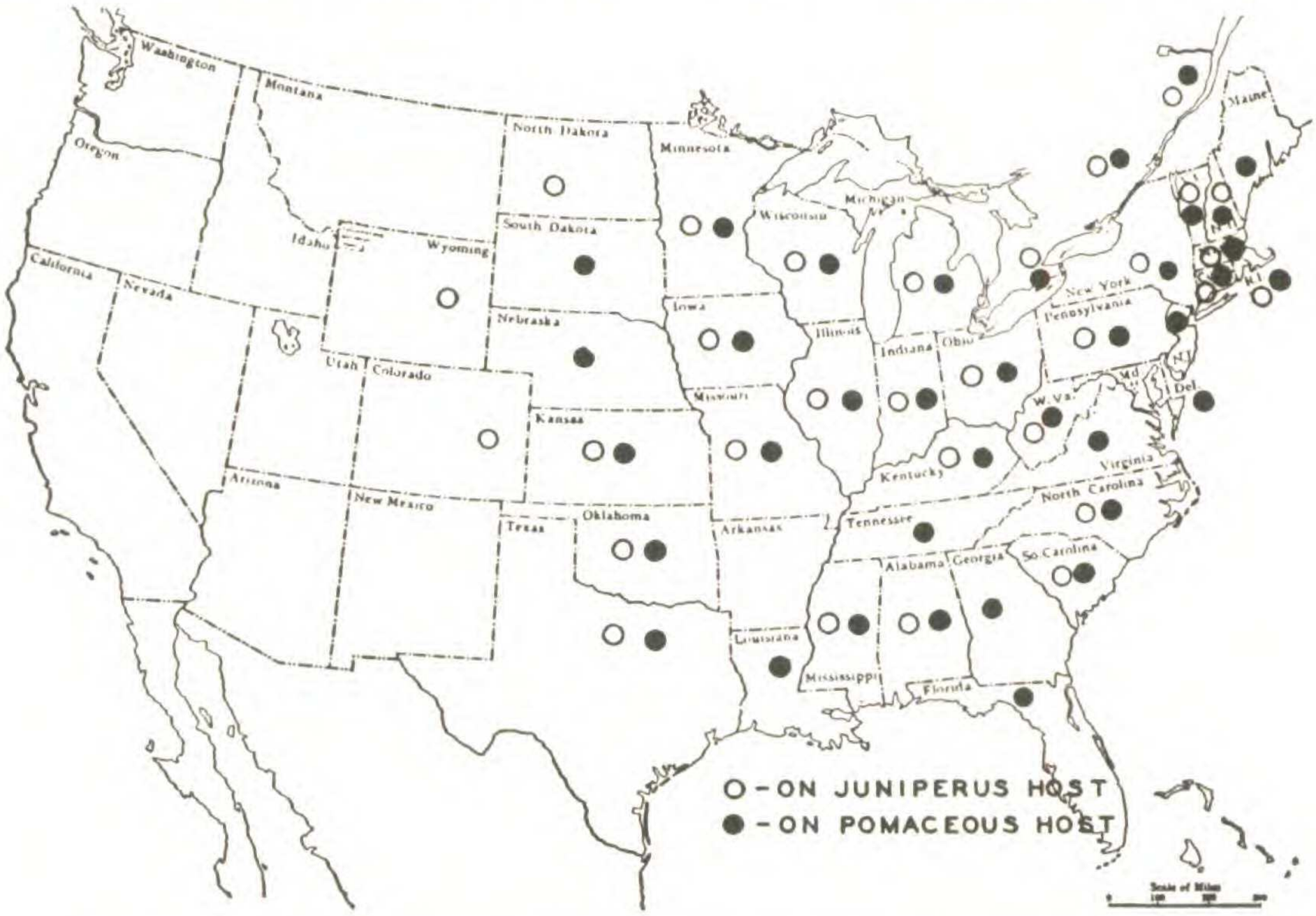

FIG. 1. DISTRIBUTION OF GYMNOSPORANGIUM GLOBOSUM BY STATES

globosum, *G. Juniperi-virginianae* and *G. clavipes*) destructive to apples in eastern New York State and report the occurrence of *G. globosum* on at least thirteen varieties of apples, with severe infection in some instances. In 1930 they (Thomas and Mills, 1930) list twenty-three varieties of apples on which the disease caused by this rust has been found. H. E. Thomas in a letter, dated Aug. 27, 1930, to the Plant Disease Reporter (Anonymous, 1930) writes that *G. globosum* was more common that year on the foliage of apples in Essex County than was *G. Juniperi-virginianae*. Another report (Anonymous, 1930) by W. S. Fields states that all the Crataegus plants of all kinds on an estate at Locust Valley, Long Island, were heavily infected. Other writers who have reported this rust in New York State include Grier (1925),

Martin (1925), Salisbury (1929), Crosby, Mills and Blauvelt (1929), Barrus, Boyd and Wood (1931) and Miller, Stevens and Wood (1933).

Severe local infections have been observed by the writer in eastern Massachusetts. Loci of infection may be found on estates where susceptible ornamental Crataegi are planted in vicinities that embrace the red cedar, and in open pastures and waste lands where wild Crataegus and Malus species are within close range of the cedar. Apparently no serious outbreaks of the diseases caused by this rust have occurred, as yet, in commercial orchards in Massachusetts.

States in which severe to moderately severe local infections have been reported include Connecticut (Clinton, 1903), Florida (Martin, 1925), Illinois (verbal report from Morton Arboretum), Michigan (Martin, 1923), Minnesota (Martin, 1920) and Ohio (Martin, 1923). Bliss (1933) states that this rust is common on apple in Indiana.

Reports would indicate that the rust is not of great economical importance in Alabama (Underwood and Earle, 1897), Iowa (Bliss, 1933), Kansas (Bartholomew, 1899), Maine (Morse and Lewis, 1910) (Miller, Stevens and Wood, 1933), New Jersey (Cook, 1917), Ontario (Connors, 1934), Wisconsin (Jones and Bartholomew, 1915) and Vermont (Anonymous, 1928). Martin (1925) reports *G. globosum* as occurring in Alaska.

IV. SYMPTOMATOLOGY OF THE DISEASES CAUSED BY G. GLOBOSUM

A. On Pomaceous Hosts

Observations were made concerning the progressive development of the symptoms and signs of the diseases caused by *G. globosum* as they appeared on the inoculations of the respective pomaceous hosts (MacLachlan, 1935). For the sake of convenience the progressive development of the symptoms and signs will be considered from both the morphological and histological viewpoints as they appeared on the foliage of *Crataegus;* modifications of these symptoms and signs as they appeared on the foliage of other hosts and on the flowers, fruit and twigs, will follow; additional observations made by other writers will be included throughout the presentation.

(a) On the foliage of *Crataegus*.

The first morphological evidence of the disease caused by this rust is the exhibition of very small light-colored areas or flecks on the upper surface of the leaf. These appear within ten to twelve days after inoculation. In rare cases no further symptoms ever occur except that the

flecks may turn brown; normally, though, these flecks become more distinct and within approximately fifteen days after inoculation (see Table I) they show as bright yellow lesions varying from one to more than ten millimeters in diameter. Sections at this stage of development reveal intercellular hyphae among the palisade and mesophyll cells (Plate 168, Fig. 5) as well as densely intertwined masses of hyphae between the epidermis and palisade layer. These masses of hyphae are the spermogonia primordia which develop to form the mature spermogonia approximately twenty-three days after inoculation (see Table I).

The mature spermogonia appear on the upper surface of the leaf, rarely on the lower, as small raised points in the center of the lesion. On rupturing the epidermis of the leaf the spermogonia exude a sticky fluid in which abundant spermatia may be found. In Plate 173, Fig. 1 may be seen a photograph of these spermatia (mag. × 545), obtained by placing a drop of the spermogonial fluid on a glass slide. Weimer (1917b) has illustrated the type of spermogonium in cross section.

TABLE I

DATA ON THE TIME OF OCCURRENCE OF THE SYMPTOMS AND SIGNS OF THE DISEASE CAUSED BY G. GLOBOSUM ON THE FOLIAGE OF CRATAEGUS[1]

	No. days after inoculation			No.
Symptoms and signs	minimum	maximum	average	species
1st appearance of lesion	13	22	15.1	38
1st exudation of spermogonial fluid	17	30	22.7	39
Spermogonia turned black	28	54	41.5	35
1st appearance of swelling	47	59	53.6	31
1st appearance of aecia	72	111	96.0	48

Exudation of the spermogonial fluid continues for approximately twenty days (see Table I), following which the spermogonia turn black and are quite conspicuous on the yellowish background (Plate 171, Fig. 1). Other than the color change of the spermogonia, little change in the lesion can be noted except for the formation, in many instances, of reddish borders around the lesions. These borders while manifested to a greater extent on some hawthorns than on others are not consistently formed even on a single host. Severe infections where more than fifty percent of the leaf area is diseased may cause yellowing of the leaf and defoliation at this stage in the development of the rust; scattered in-

[1]These data were obtained from typical representatives of the various groups of *Crataegus* inoculated with *G. globosum* Farl. in May, 1932.

fections, however, cause only minor injury to the leaf except for reduction of photosynthetic area.

Swelling on the lower surface of the leaf, opposite the spermogonia may be observed approximately ten days after the spermogonia cease exudation (see Table I). Sections of the lesions reveal that this swelling is due to both hypertrophy and hyperplasia of the mesophyll tissue, resulting in long palisade-like cells closely packed together. Hypertrophy of the leaf is confined to the area occupied by the lesion and may increase the thickness of the leaf to more than five times normal (Plate 168, Fig. 1). It is not until this stage in the development of the rust that severe leaf killing can be caused by relatively few lesions per leaf; this is especially true of vein infections (MacLachlan, 1935a).

Following the time during which hypertrophy of the lesion takes place, minute swellings may be seen on the lower surface, rarely on the upper surface, which, within two or three days time, break through the epidermis and appear as greyish acuminate cylinders about one millimeter in diameter. These are the peridia of the developing aecia and are first evident approximately ninety-six days after inoculation (see Table I). The peridia are not arranged in any definite pattern except when they occur on veins or petioles; in that case they may be arranged in two rows, one on each side of the vein or petiole (Plate 167, Fig. 4); they may develop to a length of more than six millimeters and may vary in number from one to more than fifty per lesion. Within each peridium are the aeciospores which are released by the splitting and shredding of the peridium a short distance from the distal end (Plate 171, Fig. 2). Unlike *G. Juniperi-virginianae* the peridial cells do not recurve with changes in humidity to release the aeciospores; the latter are shed through the shredded region of the peridium. High winds as well as the effect of one leaf rubbing on another have a marked influence in breaking up the peridium to release the aeciospores.

The peridial cells, as described by Kern (1911), are "broadly lanceolate in face view, 15–23 $\times$ 60–90 μ, linear-rhomboid in side view, 13–19 μ thick, outer wall about 1.5 μ thick, smooth, inner and side walls 3–5 μ thick, rather densely rugose with ridge-like papillae of varying length." As has been demonstrated by Thomas and Mills (1929b), the peridial cells are shorter and broader towards the distal end of the peridium and do not separate easily from each other.

The aeciospores are borne in chains from the base of the aecial cup and when mature are, as described by Kern (1911), "globoid or broadly ellipsoid, 15–19 $\times$ 18–25 μ, wall light chestnut brown, 1.5–2 μ thick, finely verrucose." In Plate 173, Fig. 2 may be seen a photograph of

aeciospores at the same magnification as the spermatia in Fig. 1. Two of the aeciospores (lighter color in the figure) were colorless; these colorless spores were found occasionally and proved to be viable.

Sections reveal that the aecium itself is long and narrow and deeply embedded in the hypertrophied tissue, the base of the aecium approximating that of the palisade layer of the leaf (illustrated by Weimer, 1917b). In Plate 168, Figs. 1 and 2 may be seen photographs of a longitudinal section through several aecia; the non-median sections give the appearance that the aecia begin at various levels throughout the hypertrophied tissue.

The great majority of the aeciospores are distributed by the middle of September. Remnants of the peridia may remain on the lesion until the leaves normally drop, but usually the circular pits in the aecial cups are all that can be seen at this time. Sections through the lesions after the leaves have dropped reveal very few viable aeciospores; most of those that still remain in the base of the aecium are hollow and sterile.

Insects have been observed feeding on the aeciospores during August. The peridia containing the aeciospores are eaten out first, then the insects, after gaining entrance by way of the pits into the aecial cups, proceed to break down the whole interior of the lesion.

(b) On the foliage of pomaceous hosts other than *Crataegus*.

Pyrus: The lesions on the ornamental species are smaller than those found on *Crataegus*. They vary greatly. On some host species they are very minute, being less than a millimeter in diameter and producing a few spermogonia only (e.g. *P. serrulata* Rehd.); others may exhibit two or three aecia on each lesion (e.g. *P. Balansae* Decne.); still others may exhibit lesions which are two to three millimeters in diameter with several aecia each (e.g. *P. betulaefolia* Bge.) (Plate 166, Fig. 4). On *P. betulaefolia* the red borders around the lesions are conspicuous. Stewart (1910) describes the lesions on Kieffer pear as being bright yellow and pin-head in size on June first. He states, further, that by June fifteenth the lesions had turned brown to black with conspicuous red borders on the upper surface of the leaf but without the red borders on the lower surface. He adds that a few aecia are formed on the upper side of the leaf, but most of them are on the lower side, frequently on each side of the mid-rib. Hesler and Whetzel (1917) describe the lesions as being orange-colored in June, one quarter to one half inch in diameter with red borders showing on the upper side of the leaf. In the fall the lesions turned dark on the under side of the leaf and at this time of year showed no red border.

Sorbus: The lesions in all cases were very small, rarely measuring more than one to two millimeters in diameter, with an average of three to five aecial horns per lesion (Plate I, Fig. 3). As in *Pyrus* the lesions on certain species died and turned brown shortly after the spermogonia appeared.

Malus: Inoculation on ornamental apples resulted in small lesions that rarely measured more than one to two millimeters in diameter (Plate 166, Fig. 5). Certain species exhibited spermogonia only. Crosby, Mills and Blauvelt (1929), Sherbakoff (1932) and others have reported this small type of lesion on commercial apples and refer to it as one method of distinguishing *G. Juniperi-virginianae* from *G. globosum*. Bliss (1931), by culture, obtained flecking only on nine varieties of commercial apples.

(c) On the flowers and fruit.

Infection of flowers and fruit of *Crataegus* is relatively rare when compared to the prevalence of foliar infection. All parts of the flower including pedicel, ovary, sepals, petals and even stamens may be attacked. Infections on the stamens and petals cannot persist but infections of the ovary result in either dwarfing and killing of the fruit (Plate 167, Fig. 1) or premature ripening and dropping of the fruit. Abundant spermogonia and aecia may be produced (Plate 167, Fig. 2). A cross section of a diseased fruit reveals that only a portion of the fruit is affected. In Plate 168, Fig. 3 may be seen a photograph of such a section; the diseased area is represented by the darkened strip on one side of the section. On the opposite side of the section may be seen some of the diseased area of a second lesion. As in the leaf, hypertrophied, palisade-like, densely packed host cells are evident. The fungal mycelium is intercellular and produces haustoria in the host cells. Some mycelium can be seen ramifying among the host tissue beyond the hypertrophied area.

Fruit infection of *Pyrus* was not observed in the Arnold Arboretum. Stewart (1910) reports the Kieffer pear as suffering infection from this rust at Long Island, New York. In particular he finds the diseased fruits are very small and misshapen, usually exhibiting circular black areas devoid of aecia, although a few show aecia. Hesler and Whetzel (1917) report somewhat similar symptoms on the fruit of pear.

Fruit infection of *Malus* has not been observed by the writer. Garman (1899), speaking collectively of *G. Juniperi-virginianae* and *G. globosum*, reported these rusts on the fruit of *Malus;* he found the infection to be most abundant on crab apples.

(d) On the twigs.

Infection of the twigs of *Crataegus* is relatively rare and occurs on the current year's growth only. Both spermogonia and aecia may be produced (Plate 167, Fig. 5). Similar infections of the thorns and axillary buds have also been observed (Plate 167, Fig. 3). A cross section of a diseased twig reveals that the infection is confined to the cortical region (Plate 168, Fig. 4). The same typical, densely packed, hypertrophied cells that occur in the leaves and fruit may be seen in the diseased area. The aecial cups, however, appear shorter and broader than those exhibited in diseased leaves. Several diseased twigs were tagged in the fall of 1932 to determine if the mycelium might be perennial in the twigs; such was not evident as the twigs were all dead the following spring.

B. On Juniperus Hosts

The advanced stage of the disease caused by *G. globosum* on the *Juniperus* host is expressed in the form of a woody gall attached firmly to a twig that may be more than two or three years old. This condition led early observers to conclude that infection originated in the stem. Farlow (1880) who named this fungus stated that the disease is first manifested by bursting through the stem at the point of attachment of the leaves. Kern (1911) described the gall as being caulicolous. Stewart (1915) gave an elaborate account of histological studies made, which he interpreted as showing that the gall originates from the axils of leaves and are evidently transformed axillary buds. Weimer (1917a), after examining Stewart's slides, showed that Stewart mistook an axillary bud for a gall. He (Weimer, 1917a) gave a full description of a normal juniper leaf and presented definite proof that the gall may originate in the leaf. Bliss (1933) has substantiated the results of Weimer and has illustrated stages in the progressive development of the gall on the leaf. Several brief descriptions of the mature gall as it occurs on the red cedar may be found in the literature; these descriptions are given, for the most part, as an aid in distinguishing the galls caused by *G. globosum* from those caused by *G. Juniperi-virginianae*.

To obtain a more complete picture of the progressive development of the various symptoms and signs of the disease caused by *G. globosum* on the red cedar, the writer has carried on further studies both morphologically and histologically. Material for study was obtained from a locus of infection that was isolated from other *Gymnosporangium* rusts. The symptoms and signs will be considered as they occur on the red cedar in eastern Massachusetts.

The initial manifestations of the disease caused by this rust may be found on one year old leaves of infected red cedars about the first of August. The exhibition of a yellowish chlorotic zone or band on the leaf is closely followed by the formation of a slightly raised area on the inner (upper) surface of the leaf; the lifting of the epidermis over the point of infection is due to the development of a very young gall underneath.

Longitudinal splits in the epidermis immediately over the raised area allow the young gall to emerge. On long subulate leaves the gall may occur at any place throughout the length of the leaf; in Plate 169, Figs. 2 and 3, may be seen young galls developing near the tip, in the middle and near the base of the leaf. The usual type of leaf and young gall that is found is illustrated in Plate 169, Fig. 1; the leaves are short and the gall must of necessity be brought in close contact with the stem, a phenomenon which led early investigators to conclude that the gall originates in the stem at the base of the leaf.

A longitudinal section of a portion of a twig bearing two normal leaves as well as a diseased leaf, at approximately the same stage of gall development that is shown in Plate 169, Fig. 1, is illustrated in Plate 170, Fig. 1. A comparison of the two normal leaves with the diseased leaf reveals marked hypertrophy and hyperplasia of the mesophyll of the latter. Distortion of the vascular system may also be seen. Figs. 4 and 5 of Plate 170 exemplify further the distortion of the vascular elements. In Fig. 4 parenchymatous cells may be seen separating the vascular elements. In Figs. 2 and 3 a corky layer, several cells thick and completely surrounding the portion of the gall not in contact with the twig, may be seen. This corky layer usually cuts off the tip of the leaf; it is of interest to note in Figs. 3 and 4 the sharp severing of the vascular strand. The tip of the leaf that is cut off together with the mycelium of the rust that may be found in it dies; remnants of this dead portion may be found on the gall for two or three years afterwards. Intercellular mycelium is abundant throughout the gall tissue but haustoria were not observed at this stage.

The gall as seen in late autumn and winter is smooth, shiny and mahogany red in color, rarely exceeding a diameter of half a centimeter. This is in striking contrast to the larger, greenish and more or less convoluted gall of *G. Juniperi-virginianae*.

About the last of March of the following spring light orange colored markings may be seen over the surface of the gall. These markings represent early stages in the rupturing of the corky epidermis and are caused by the underlying masses of hyphae—the telia primordia—from

which the teliospores arise. Progressive stages in the maturation of the teliospores from this dense hyphal layer are shown in Plate 171, Figs. 5, 6 and 7. Dodge (1918) has published an extensive cytological study of the development of the teliospores and has illustrated progressive stages of their maturation. The teliospores mature progressively from the centre towards the periphery of the layer of telia primordia. The corky epidermis is ruptured over the maturing teliospores and the latter emerge as a cinnamon-brown, pulvinate mass (Plate 171, Fig. 4). The irregular outline of this rupture readily distinguishes the galls caused by this rust from those caused by *G. Juniperi-virginianae* since the telial sori of the latter emerge through circular openings. The teliospores are borne on pedicels and the extension of these pedicels, as the teliospores progressively mature from the centre towards the periphery of the basal layer of mycelium, enlarges the sorus to form a tongue or wedge-shaped structure, the centre of which may be hollow. This cavity is due to the developing teliospores around the peripheral regions of the sorus; these push up the mature teliospores in the centre and as a result the pedicels of the latter teliospores are broken off.

A mature telial sorus, if kept dry, is a tongue-like or wedge-shaped flange 1–3 mm. broad by 2–5 mm. long at the base and 6–12 mm. high. A section through such a sorus reveals a dense cinnamon-brown layer of teliospores, several spores thick, over the surface. Under the spore layer is a dense white mass composed entirely of long pedicels, one for each teliospore. The peripheral pedicels still remain attached to the layer of mycelium from which they arose; while the pedicels in the central region, which have been broken off from the layer of mycelium from which they arose, are suspended to form a conical amphitheatre-like cavity in the centre.

The teliospores are typically two-celled and are, as described by Kern (1911), "ellipsoid, 16–21 × 37–48 μ somewhat narrowed above and below, slightly constricted at the septum, wall pale cinnamon-brown, 1–2 μ thick; pores 2 in each cell, near the septum." Pammel (1905) has described one-celled teliospores. No single-celled teliospores have been observed by the writer; three- and four-celled teliospores, however, as well as four-celled teliospores with one cell abortive have been found (Plate 173, Fig. 3).

Studies with respect to the nature of the pedicels of the teliospores and the effect of wetting will be considered later (page 16).

Wetting of the telial sori by rains in May causes them to expand to many times their original size. Progressive stages in the expansion of sori on galls as well as on the small infections that take place on the long

subulate leaves are illustrated in Plate 172. The hygroscopical nature of the pedicels (see page 16) is responsible for this expansion. If only a portion of the teliospores are mature at the time of wetting, the telial mass may, on drying, shrink to somewhat of its original shape and size. If, however, all the teliospores are mature at the time of original wetting and the humidity remains high for a sufficient length of time to fully expand the telial mass, the latter, on drying, will shrink to an amorphous sheet or strand and later fall away from the gall. Under favorable weather conditions practically all the teliospores are mature within three to five weeks from the time of their initial appearance.

Germination of the teliospores results in the production of basidiospores. Progressive stages in the production of the latter are shown in Plate 174, Fig. 1. The basidiospores may be seen as a powdery, somewhat velvety coating over the surface of the expanded telial sorus and are evident about five or six hours after the expansion takes place. These basidiospores will drop from the gall of their own accord but are usually carried away by the air currents. The basidiospores are ovate, flattened on one side, slightly tapering towards the end of attachment to the sterigmata; thin-walled (less than 1 μ thick); protoplasm light orange-yellow in color containing numerous oil droplets which tend to aggregate (Plate 173, Fig. 4); size, 12–16 (average, 13.7 μ) × 7–11 (average, 8.9 μ). Under favorable conditions they germinate immediately after their formation to form relatively long germ tubes (Plate 174, Fig. 4). If the spores are allowed to dry shortly after the germ tubes begin to develop, small secondary spores may be formed (Fig. 5).

As a rule, the majority of the teliospores have germinated by May 25. The dried up remnants of pedicels and empty teliospores blow away leaving a smooth orange colored scar bordered by the lacerated edges of the broken corky epidermis. This scar soon turns greyish brown and may persist on the gall surface during the remainder of the life of the gall (Plate 171, Fig. 4). Cross sections through the gall reveal that the tissue immediately underlying this scar dies and a corky layer develops, segregating this dead area from the remaining living tissue of the gall (Plate 170, Fig. 5).

The gall continues growth during the summer causing distortion of the neighboring twigs (Plate 169, Fig. 5) and in some cases killing of the twig beyond the gall (Plate 169, Figs. 4 and 6). The original single vascular strand of the leaf now shows as a many-branched structure (Plate 170, Fig. 5). New growth beyond the gall as well as the natural dropping of the older leaves and the formation of a corky epidermis on the twig surrounding the gall give the appearance that the gall originated

in the stem rather than in the leaf. This illusion is further substantiated by the tendency of the gall to become woody in appearance as it grows older (Plate 169, Fig. 7).

The gall is commonly referred to as being perennial in nature. For how many years it will live and produce teliospores is not known; in Plate 169, Fig. 7 is shown a gall that produced teliospores for three consecutive years. The telial sori are formed each year between the scars where teliospores were produced the previous years (Plate 171, Fig. 4). It is doubtful that many of the galls persist for more than three or four years, because, by that time the surface of a gall would be completely covered with dead tissue resulting from the formation of the telial sori. Long subulate leaves on which infection takes place usually drop after the first crop of spores; these infections, then, can persist for one year only.

V. FACTORS AFFECTING SPORE GERMINATION

A. The Time Factor

The teliospores exhibit a low and inconsistent percentage of germination when tested immediately after their appearance on the gall. If twigs bearing galls on which teliospores are just emerging are placed in water in the greenhouse, abundant germination can be obtained, regularly, in a week to ten days time. The maturation period of the teliospores is longer, depending on the weather conditions, when the galls bearing them are left in the field. Normally, the teliospores will all have germinated within six to eight weeks after their formation. Under low temperature conditions, however, they have the potential ability to remain viable for a much longer time; in Plate 175, Fig. 3 may be seen severe infection obtained on leaves of *Crataegus Jonesae* Sarg. using, as inoculum, teliospores that had been kept in the refrigerator for one year.

The basidiospores have the ability to germinate immediately after their formation. The length of time that they will live is still a question; basidiospores of *G. Juniperi-virginianae* will live for many days above a humidity of 25% and below a temperature of 25° C. (MacLachlan, 1935b).

Aeciospores will not germinate to the extent of more than 2 or 3% at the time of their formation. Nevertheless, if hawthorn leaves bearing aecia are collected in August before the aeciospores are shed and placed in a refrigerator at 0° C., abundant germination can be obtained within a month's time and by the first week in October more than 80% germination can be obtained consistently. By keeping the infected leaves at this temperature, the same high degree of germination can be

obtained until the first of March of the following year; after this date, however, the percentage of germination that can be obtained falls off rapidly. Whether the infected leaves are kept in a dry container or a moist chamber makes no difference in the percentage germination obtained. It may be noted that on one occasion more than 75% germination was obtained in August, 1932, using aeciospores taken from unbroken peridia of an infected leaf of *Crataegus shirleyensis* Sarg.

B. The Humidity Factor

Tests in the laboratory revealed that none of the three spore forms would germinate, even in saturated humidity (spores placed on cover glasses over water), unless in direct contact with a drop of water. Excess water, however, causes irregularity in the percentage germination; basidiospores, especially, show a very low percentage of germination when immersed in large drops of water; under the same conditions aeciospores are not consistent in the percentage of germination that may take place; teliospores germinate in excess water but instead of producing basidiospores the promycelia grow to great lengths, presumably to come in contact with the air, and may exhibit long side tubes (Plate 174, Fig. 3), or break up to form elongated spore-like bodies (Plate 174, Fig. 2). Farlow (1880) reported a type of spore, similar to that shown in Fig. 2, which he observed when the telial masses were quickly dried after moistening. He also found that, on remoistening, these spore-like bodies would send out germ-tubes similar to those of normal basidiospores.

In the laboratory, optimum humidity conditions for germination of any of the three spore forms was obtained by placing the spores on a cover glass and inverting the latter on a Van Tieghem cell in a petri-plate lined with wet filter paper; enough water of condensation would accumulate on the lower (spore) surface of the cover glass to give optimum humidity conditions for germination. In the field, very satisfactory humidity conditions for infecting the pomaceous host were obtained by painting the leaves with an aqueous suspension of the teliospores and enclosing the inoculated twig in a celluloid cylinder, the ends of which were plugged with moist sphagnum.

C. The Temperature Factor

Temperature has a marked effect on the percentage of germination that may be obtained. Miller (1932) found 24° C. to be the optimum temperature for the germination of teliospores and aeciospores, and 16° C. the optimum for basidiospores. In Table II may be found data

TABLE II

DATA ON THE PERCENTAGE GERMINATION OF THE SPORES OF G. GLOBOSUM[1] WHEN SUBJECTED TO DIFFERENT TEMPERATURES[1]

Temp. °C.	Percentage germination of teliospores	basidiospores	aeciospores
2	0.0	—	0.0
5	0.3	0.0	5.2
7	0.7	7.2	8.2
10	80.4	68.7	11.3
15	90.9	79.2	70.2
20	96.3	83.9	88.7
25	98.6	63.1	60.7
30	59.8	14.5	35.3
35	0.9	0.9	0.6
38	0.0	0.0	0.0

on the percentage germination of the three spore forms when subjected to various temperatures; these data have been plotted in Fig. 2. Examination of Fig. 2 reveals that: (1) the teliospores have a very wide range of temperature within which more than 80% germination may take place, (2) the corresponding curve for basidiospore germination has the same general contour, except at a lower level, (3) the aeciospores have a narrower range of temperature within which a high percentage of germination may take place, the optimum being around 20° C., (4) below a temperature of 10° C. and above a temperature of 30° C. the percentage germination of all three spore forms drops off to almost zero.

VI. THE NATURE OF THE TELIAL SORUS

Reference was made under SYMPTOMATOLOGY (page 12) to the marked expansion of the telial excrescences on the galls during wet weather. In Plate 172 may be seen photographs of progressive stages in the expansion of these telia. Further studies were made with respect to this phenomenon.

It was found that the pedicels of the teliospores, when wetted, are responsible for this expansion process. No estimation has been made with respect to the number of teliospores and the corresponding number of pedicels that may exist in a single telium, yet, they must number in the millions. Crowell (1934) has given estimations which indicate that four to five million teliospores may exist in a telium of *G. Juniperi-*

[1]1500 spores were counted in each sample. Counts were made 24 hours after the cultures were set up.

virginianae; the number that may exist in a telium of *G. globosum* would be as many or more as the telia are characteristically larger in the latter rust. The pedicels, as well as being numerous, are relatively long; some idea of their length may be obtained in Plate 175, Fig. 5. Under strong light and on a dark background they appear white and opaque; they do not stain readily in aqueous methylene blue. In Plate 175, Fig. 4 may be seen a photograph of a pedicel that was immersed in 25% alcoholic

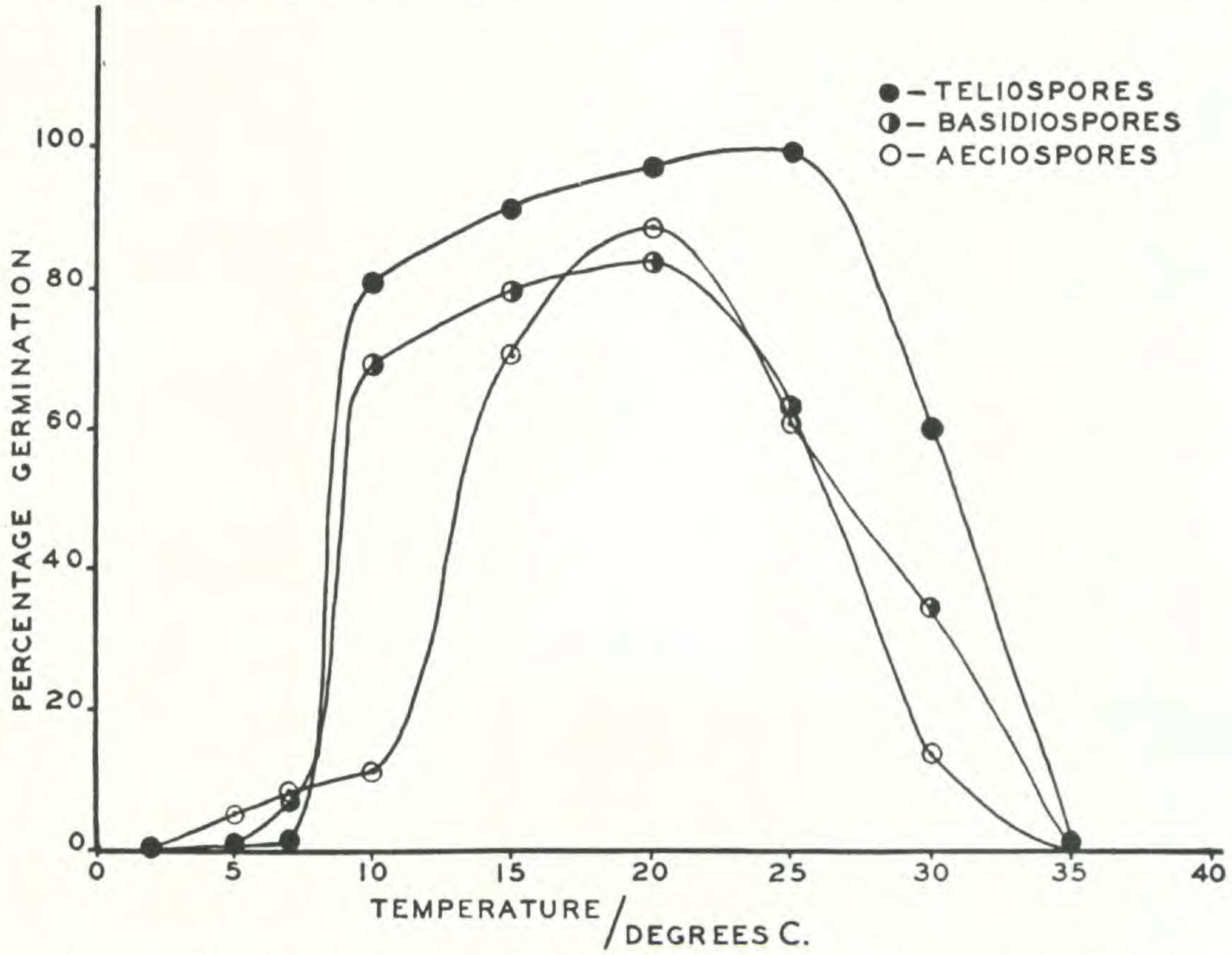

FIG. 2. THE INFLUENCE OF TEMPERATURE ON THE PERCENTAGE GERMINATION THAT MAY BE OBTAINED FOR THE THREE SPORE FORMS.

methylene blue for fifteen hours; the pedicel itself did not stain to any extent but the stain was absorbed in the centre of the pedicel indicating the presence of a lumen. The hygroscopic nature of the pedicel is illustrated in Plate 175, Figs. 1 and 2. The pedicels represented in Fig. 1 were subjected to 95% alcohol for twelve hours while those represented in Fig. 2 were placed in water for the same length of time. The two pictures are at different magnifications but when one compares the diameter of the pedicel with the size of the teliospore which it subtends, it is quite evident that the pedicels are capable of considerable expansion when wet; Fig. 1 illustrates the pedicel as it would appear when the telium is dry while Fig. 2 illustrates the pedicel as it would appear during

rainy weather in May. In Fig. 2 may also be seen small air bubbles existing in the region that was once the lumen but is now filled by the expanded wall of the pedicel. On the other hand, as may be seen in Fig. 1, the lumen is relatively wide with respect to the diameter of the desiccated pedicel. Taking into consideration, then, the number of pedicels present in a telium as well as their length and the diameter they may attain when wet, one can readily see how a telium may expand to the size that is so common on infected cedars during rainy periods in May.

VII. INFECTION OF THE RED CEDAR

A. Time of Infection

The actual time of year that infection of the red cedar by the aeciospores takes place is still conjectural. About 2% of the aeciospores will germinate at the time of their dispersal in late August and when one realizes the large number of aeciospores that may be released from infected pomaceous hosts it can be presumed that possibly some infection may take place immediately. Nevertheless, the fact that more than 80% germination can be obtained after the fresh aeciospores have been subjected to 0° C. for about six weeks time (cf. page 14) would indicate that both time and low temperature are involved in the maturation of the aeciospore. This would indicate that infection takes place in late fall after frosts have occurred. The fact that a high percentage of germination can be obtained in March of the following spring, using aeciospores that have been kept at 0° C. during the interim, would not necessarily mean that infection takes place in the spring; if the aeciospores are mature in late fall, in all probability they will have all germinated long before March.

B. Infection Process

Having access to an abundance of aeciospores which would germinate readily, attempts were made to trace the method by which infection of the red cedar by this rust takes place.

Leaves of the current year's growth were removed from potted red cedars, washed carefully and dusted on the stomatal (inner and upper) surface with aeciospores. The inoculated leaves were then inverted and placed on moist filter-paper in petri-plates. Examination 24 hours later revealed abundant germination of the aeciospores; the germ tubes were long and slender and sometimes spiralled; the protoplasm had all migrated to the end of the germ tubes. Within 12 hours more, the ends of the germ tubes had enlarged to form irregular and sometimes convoluted

haustoria-like formations. On one occasion the germ tube was observed to bend at a sharp right angle to form the haustoria-like structure over a stoma; however, no consistent orientation of the germ tubes with respect to the location of stomata could be observed. The haustoria-like structures that happened to form over stomata were smaller than those that formed on the inter-stomatal regions of the leaf epidermis. Two samples of about fifty leaves each were removed from the culture and killed for embedding thirty-six and fifty-two hours, respectively, from the time of inoculation. These were sectioned (both transversely and longitudinally), stained with safranin and light green and examined under the high power of the microscope in hopes of observing the method of penetration.

The length of the germ tubes made it impossible to obtain even a series of sections that showed an aeciospore connected to its germ tube throughout its entire length. This led to a difficulty in the identification of germ tubes that were observed entering the leaf and consequently the results obtained can only be considered as indications and not conclusive proof. The characteristic intercellular mycelium of *G. globosum* was found around the mesophyll cells in the immediate vicinity of the stomata. Hyphae of the same diameter as the germ tubes were found passing through the stomata; in some instances three or four passed through the same stoma. Some of the haustoria-like structures were found to be still clinging to the surface of the leaf but no penetration from them was observed.

Whether the germ tubes that were observed passing through the stomata were those of *G. globosum* or of some other fungus that happened to be mixed with the aeciospores cannot be said for certain. It is quite safe to say, however, that the rust gains entrance through the upper epidermis of the leaf on which the stomata exist, as the characteristic intercellular hyphae of this rust were found around the mesophyll cells immediately under the stomata. Further studies on this problem will be carried out by the writer.

VIII. SUMMARY

At least thirteen names have been given to the rust now known as *Gymnosporangium globosum* Farl. Of these the authentic names for the III and O & I stages of the rust stand as *Gymnosporangium globosum* Farl. and *Roestelia globosa* Shear, respectively. The name *Gymnosporangium globosum* Farl. is now accepted as referring to either stages of the rust.

Gymnosporangium globosum is confined in its range to the eastern and central parts of the United States and to the southern parts of Ontario and Quebec. The rust is increasing in prevalence and in localized areas is causing great damage to both the ornamental and orchard trees; this is especially true in eastern New York State.

The diseases caused by *G. globosum* occur on at least ten genera of the Pomoideae and on at least three species of *Juniperus*. The symptoms and signs of the diseases caused by this rust may be found on the foliage and to a lesser extent on the flowers, fruit and twigs of the pomaceous hosts and on the foliage and twigs of the Juniperus hosts. The progressive development of these symptoms and signs as they occur on the aforementioned organs of the respective hosts have been described and illustrated.

The factors of time, humidity and temperature have been considered with respect to the percentage germination of the teliospores, basidiospores and aeciospores that may be obtained. The age of the three spore forms as well as the temperature to which they are subjected have a marked effect on the percentage germination that can be obtained. The amount of water present also modifies the percentage germination that can be obtained as well as the type of germ tubes that may be exhibited.

All evidences indicate that infection of the red cedar by *G. globosum* occurs, primarily, in late autumn. From studies of the infection process it would appear that infection of this host takes place through the upper and stomatal surfaces of the leaves.

IX. ACKNOWLEDGMENTS

To Professor J. H. Faull, who afforded the writer the opportunity to study this problem and for guidance and supervision during the pursuance of its investigation, sincere appreciation is expressed.

The writer is also grateful to Dr. Ivan H. Crowell for many helpful suggestions and to Mr. Francis M. Bryant for technical assistance.

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XI. EXPLANATION OF PLATES

PLATE 166

Aecial stage of *G. globosum* on the foliage of pomaceous hosts. Unless signified inoculations were artificial.

Fig. 1. On *Crataegomespilus grandiflora* Bean. Note leaf killing.
Fig. 2. On *Crataegus prunifolia* (Marsh.) Persoon.
Fig. 3. On *Sorbus americana* Marsh. The lesions are very small.
Fig. 4. On *Pyrus betulaefolia* Bge.
Fig. 5. On *Malus* sp. (Natural infection.)

PLATE 167

Aecial stage of *G. globosum* on various parts of Crataegus hosts. (Artificial inoculations.)

Fig. 1. Dwarfing and killing of fruit.
Fig. 2. Abundant aecia produced on basal portion of a fruit.
Fig. 3. On a terminal bud that was forming in the axil of a leaf.
Fig. 4. On the petiole of a leaf.
Fig. 5. On twigs of the current year's growth.

PLATE 168

Histological sections of *Crataegus* showing diseased areas.

Fig. 1. Cross section through a diseased Crataegus leaf showing the amount of hypertrophy that may occur in the lesion. Longitudinal sections through various portions of aecia may be seen also.
Fig. 2. An enlarged portion of a lesion similar to that shown in Fig. 1. Hypertrophy of the mesophyll has resulted in palisade-like, densely packed, cells in which the chloroplasts have disappeared. The section also shows a near-median section of an aecium.
Fig. 3. Section through a diseased Crataegus fruit. Hypertrophied area of the lesion shows as a dark strip in the photograph. This area is composed of densely packed, palisade-like, cells similar to those which occur in the foliar lesions.

Fig. 4. Cross section through a lesion on a Crataegus twig. Hypertrophy and intercellular mycelium are confined to cortical regions. The aecial cups are characteristically shorter and broader than those found on the foliage.
Fig. 5. Intercellular mycelium in the mesophyll of a diseased Crataegus leaf under a spermogonium before hypertrophy has set in.

Plate 169

Galls of *G. globosum* in various stages on leaves and twigs of Juniperus host (Red-cedar).

Fig. 1. The usual type of young galls that may be seen breaking through the upper surfaces of leaves about September 1st.
Figs. 2 and 3. Young galls at a similar stage of development that is shown in Fig. 1, in long subulate leaves.
Fig. 4. A one-year-old gall that has caused the death of the twig beyond it.
Fig. 5. A two-year-old gall that is causing distortion of the twigs.
Fig. 6. A two-year-old gall that has killed the twig beyond the point of infection. The dead portion of the twig has fallen away. (See Fig. 4.)
Fig. 7. An old gall (3 or 4 years) that gives the appearance of having originated in the stem. The infection that resulted in this gall took place when the twig bearing it was young and covered with green leaves.

Plate 170

Histological sections of *Juniperus* infected with *G. globosum.*

Fig. 1. Longitudinal section through a twig bearing two normal leaves and an infected leaf. Note hypertrophy of the infected leaf as well as the distortion of the single strand of vascular elements.
Fig. 2. A later stage than that shown in Fig. 1. A corky layer several cells thick has set in that surrounds the gall and cuts off the tip of the leaf. The tip of the leaf that is cut off together with the rust mycelium that may be found in it dies but may persist in the gall for two or three years.
Fig. 3. Enlarged portion of gall similar to that shown in Fig. 2. Note sharp severing of the vascular strand by the corky layer.
Fig. 4. Enlarged portion of section shown in Fig. 3. Note parenchyma-like cells that are forming within the vascular strand splitting up the latter.
Fig. 5. A section through a two-year-old gall. The splitting up of the single vascular strand of the original leaf has now resulted in a many-branched structure. On the upper side may be seen the dead portion on which a telial sorus was produced the previous spring; a corky layer had set in, segregating the dead portion from the remainder of the gall.

Plate 171

Fruiting structure of *G. globosum* on *Crataegus* (Figs. 1 and 2) and *Juniperus* (Figs. 3–7).

Fig. 1. Spermogonia in the center of a foliar lesion; they have turned black and are quite conspicuous on the yellow background of the lesion.
Fig. 2. Typical aecia on the foliage. Note method of shredding of the peridial cells for the release of the aeciospores.

Fig. 3. Telial sori breaking through a young gall for the first time.
Fig. 4. Telial sori breaking through the epidermis of the gall shown in Plate 169, Fig. 7. The irregular method by which the sori rupture the epidermis readily distinguishes the galls caused by *G. globosum* from those caused by *G. Juniperi-virginianae* in which circular pit-like openings are formed. Note also the scars between the sori; within the area occupied by these scars telial sori occurred in previous years.
Figs. 5, 6 and 7. Progressive stages in the development of the layer of telia primordia that form under the corky epidermis of a gall in March. The corky layer is ruptured about the time that the teliospores are formed.

PLATE 172

Gelatinization of the telial excrescences.

Figs. 1 and 2. Before and after wetting of two infections on a long subulate leaf. No gall formation was evident on this diseased leaf.
Figs. 3, 4 and 5. Progressive stages in gelatinization of three telial sori on a single one-year-old gall.
Figs. 6 and 7. Before and after wetting of a single infection on a long subulate leaf. Note the formation of a gall on this leaf.
Fig. 8. Gelatinization of the usual type of gall that is found.

PLATE 173

Spore forms of *G. globosum* (Magnification × 545).

Fig. 1. Spermatia.
Fig. 2. Aeciospores. Two of the aeciospores shown in this picture were colorless; these spores proved to be viable.
Fig. 3. Teliospores. The usual type are two-celled. Occasionally three- and four-celled spores may be found. Note the four-celled teliospore with one cell abortive.
Fig. 4. Basidiospores. Note the conspicuous yellow oil droplets.

PLATE 174

Germinating spores of *G. globosum*.

Fig. 1. Progressive stages in the germination of a teliospore to the formation of basidiospores. (A portion of the promycelium of the upper cell was out of focus and does not show in the last picture.)
Fig. 2. Two promycelia of a teliospore that have broken up to form spore-like bodies instead of the normal production of basidiospores. Farlow (1880) observed these spore-like bodies germinating. They occur frequently if the teliospore is in too much water.
Fig. 3. A promycelium of a teliospore that has produced long projections instead of basidiospores. This as well occurs frequently if the teliospore is in too much water.
Fig. 4. Germinating basidiospores.
Fig. 5. A germinated basidiospore that has produced a secondary spore. Secondary spores are frequently found when the basidiospores are allowed to dry shortly after germination begins.
Fig. 6. A germinated basidiospore that has produced a side branch.
Fig. 7. Germinating aeciospores.

GYMNOSPORANGIUM GLOBOSUM Farl.